STUDY OF PROGNOSTIC FACTORS IN ORAL MALIGNANCY

Dissertation submitted in partial fulfillment of

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General surgery

Tirunelveli Medical College, Tirunelveli

The Tamilnadu Dr MGR Medical University,

Chennai, Tamilnadu.

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STUDY OF PROGNOSTIC FACTORS IN ORAL MALIGNANCY

CERTIFICATE

This is to certify that the work entitled “STUDY OF PROGNOSTIC FACTORS IN ORAL MALIGNANCY” which is being submitted for M.S. General Surgery, is a bonafide work of Dr. MENANDER. M, Post Graduate student at Department of General Surgery, Tirunelveli Medical College, Tirunelveli.

DEAN

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CERTIFICATE

Certified that consolidated dissertation "STUDY OF PROGNOSTIC FACTORS IN ORAL MALIGNANCY", presented here by Dr. Menander M, is based on bonafide cases investigated and studied the candidate himself in the wards of Tirunelveli medical college, Tirunelveli.

Prof Dr S Soundararajan,

Professor and Head,

Department of General Surgery,

Tirunelveli Medical College,

Date: Tirunelveli.

Dr. S. SOUNDARARAJAN, M.S.
PROFESSOR & H.O.D.
DEPARTMENT OF SURGERY
TIRUNELVELI MEDICAL COLLEGE
TIRUNELVELI - 11

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TIRUNELVELI

CERTIFICATE

Certified that Dr Menander M, has worked on the dissertation "STUDY OF PROGNOSTIC FACTORS IN ORAL MALIGNANCY", under my guidance and supervision. The consolidated report presented here is based on bonafide cases treated in Tirunelveli medical college, Tirunelveli. The observations and conclusions made by the candidate are his own and have been verified by me.

Guide:

Dr R. Maheswari MS,

Associate Professor,

Dept of General Surgery,

Tirunelveli Medical College,

Tirunelveli.

CIVIL SURGEON
Tirunelveli Medical College Hospital
TIRUNELVELI-11.

DECLARATION

This is a consolidated report on "STUDY OF PROGNOSTIC FACTORS IN ORAL MALIGNANCY" based on patients with oral malignancies admitted in Tirunelveli Medical College Hospital, Tirunelveli during the period from February, 2011 to August, 2012.

This is submitted to the Tamilnadu Dr M.G.R. Medical University in partial fulfillment of the rules and regulations for the M.S degree examination in General Surgery.

Tirunelveli Medical College Hospital,

Tirunelveli.

Menander M

ETHICS COMMITTEE APPROVAL

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Menander M

Menander M

STUDY OF PROGNOSTIC FACTORS IN ORAL MALIGNANCY

Dissertation submitted in partial fulfillment of

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ABBREVIATIONS AND ACRONYMS

AIDS	Acquired Immunodeficiency Syndrome
AJCC	American Joint Committee on Cancer
AUC	Area Under Curve
CR	Complete Response
CT	Computed Tomography
	Chemotherapy
DNA	Deoxyribose Nucleic Acid
ECOG	Eastern Co operative Oncology Group
EGFR	Epidermal Growth Factor Receptor
5 FU	5 Fluorouracil
FNAC	Fine Needle Aspiration Cytology
HPV	Human Papilloma Virus
ICMR	Indian Council of Medical Research
IMRT	Intensity Modulated Radiotherapy
MRI	Magnetic Resonance Imaging
PET	Positron Emission Tomography
PD	Progressive Disease

PR Partial Response

RECIST Response Evaluation Criteria In Solid Tumours

RMT Retromolar Trigone

RT Radiotherapy

SCC Squamous cell carcinoma

STD Sexually Transmitted Diseases

TNM Primary Tumor, Regional Nodal Metastases, Distant Metastasis

TvMCH Tirunelveli Medical College

UV Ultraviolet

WHO World Health Organisation

GLOSSARY

Quid Betal nut is chewed in combination with lime and cured tobacco as a mixture

Retromolar Trigone Retromolar trigone is represented by tissue posterior to the posterior inferior alveolar ridge and ascends over the inner surface of the ramus of the mandible

Sideropenic dysphagia Plummer Vinson syndrome. It's a triad of Dysphagia [Esophageal webs], Iron Deficiency Anemia and Glossitis.

INTRODUCTION

INTRODUCTION

Squamous cell carcinoma is an aggressive neoplasm ranking sixth worldwide. Oral cavity malignancy is of squamous in origin and it accounts for 95% of head and neck malignancies.[1]Most cases of oral malignancies are diagnosed at an advanced stage in the Indian subcontinent. Incidence of oral malignancies is more common in the Indian setup when compared to the western world. In developed countries, the incidence among the annually diagnosed neoplasms is 3% compared to Asia and India (developing countries) where it represents almost 30%.[2]Various clinical and histopathological factors have been addressed in oral malignancies.

The American Joint Committee on Cancer (AJCC) staging system classified head and neck malignancies into six major groups: lip and oral cavity, pharynx, larynx, nasal cavity and paranasal sinuses, major salivary glands, and thyroid. Male preponderance in aerodigestive tract malignancies has been noted significantly, but male-to-female ratio is steadily decreasing because of the increased incidence of female smokers. The highest incidence areas of France, Hong Kong, India, Spain, Italy, and Brazil, as well as in U.S. blacks incidence in males exceed 30 per 100,000. The highest female rate greater than 10 per 100,000 is found in association with chewing of betel quid and tobacco in India.[3] In India, cancer is the second most common disease responsible for maximum mortality with about 0.3 million deaths per year.[4] Mouth and oropharynx is the most common malignancy in males and third most common malignancy in females following cervix and breast as per ICMR 2004.[5]

In oral mucosa, a continuous spectrum is observed beginning with preinvasive lesions, ending with invasive carcinomas and metastasis.[1] Tobacco abuse and alcoholism are the two most common risk factors which are modifiable in the development of head and neck malignancies and oral malignancies in particular.[6] The other risk factors associated are environmental UV

light, poor nutrition, HPV infection, immunosuppression, mechanical irritation, thermal injury, and chemical exposure, chronic infection with syphilis and Plummer-Vinson syndrome.[6,7] Mechanical irritation, thermal injury and chemical exposure have been addressed after noting their association with pipe smoking. The risk factors are synergistic and not only additive, especially tobacco use and alcoholism[6]. Chronic exposure to risk factors lead to precursor/preinvasive lesions by two hit process- DNA damage and the ability of mutated cells to progress through the rest of the cell cycle, leading to invasive lesions.[6]

Various clinical and histopathological factors have been addressed in oral malignancies to help in prognosticating the disease process to aid in formulating treatment protocol. Hence this study has been undertaken to evaluate the factors in prognosticating oral malignancies.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Majority of the neoplasms of the head and neck is represented by Squamous cell carcinoma accounting to about 95%.[1]Hence the diagnosis and treatment of lesions in the head and neck including oral cavity require a systematic approach.[6] The American Joint Committee on Cancer (AJCC) staging system classified head and neck malignancies into six major groups: lip and oral cavity, pharynx, larynx, nasal cavity and paranasal sinuses, major salivary glands, and thyroid.[5]

History

The first clinical description of oral cancer was by Sushruta almost 2500 years back in his "Sushruta Samhita".[7] In 1902, Niblock[8] has reported high incidence of oral cancer, particularly cancer buccal mucosa in southern parts of India and indicated that this was due to the frequent habit of chewing pan and tobacco and the disease site most often found at the site of long standing contact of quid to mucosa. Balram[8], in the same year, expressed the view that slaked lime as the main source of carcinogen in buccal mucosal cancer.

Fells[8] reported a series of 390 cases of malignant diseases, reported 346 oral cancers, with buccal mucosal cancer constituting 83% of it. Abba[8] incriminated that the etiological agent of oral cancer to be tobacco in the year 1915. The development of premalignant lesions which later develop into cancer with prolonged and continuous use of tobacco was suggested by Bloodgood[8] in 1921. Orr[9] in the year 1933, attributed shell lime use and nutritional deficiencies as the cause of cancer, after observing the fact that oral cancer was less in incidence in northern India when compared to the south. The high incidence of palatal cancer in people with habit of smoking of chutta with burning end inside the mouth [reverse smoking] was reported by Kini and Rao in 1937.[8] Ackermann, in the year 1948 suggested verrucous carcinoma was a distinct entity of squamous carcinoma.[10,11]

The first WHO accepted classification for oral and oropharyngeal tumours was given in the year 1971 by Wahi P.N., Cohen B, Luthra V.K., and Torloni H.[12] The recent WHO classification of tumours of oral cavity was given in the year 2003 and it broadly classifies into the following types

Malignant epithelial tumors

Epithelial precursor lesions

Benign epithelial tumors

Salivary gland tumors

Soft tissue tumors

Hematolymphoid tumors

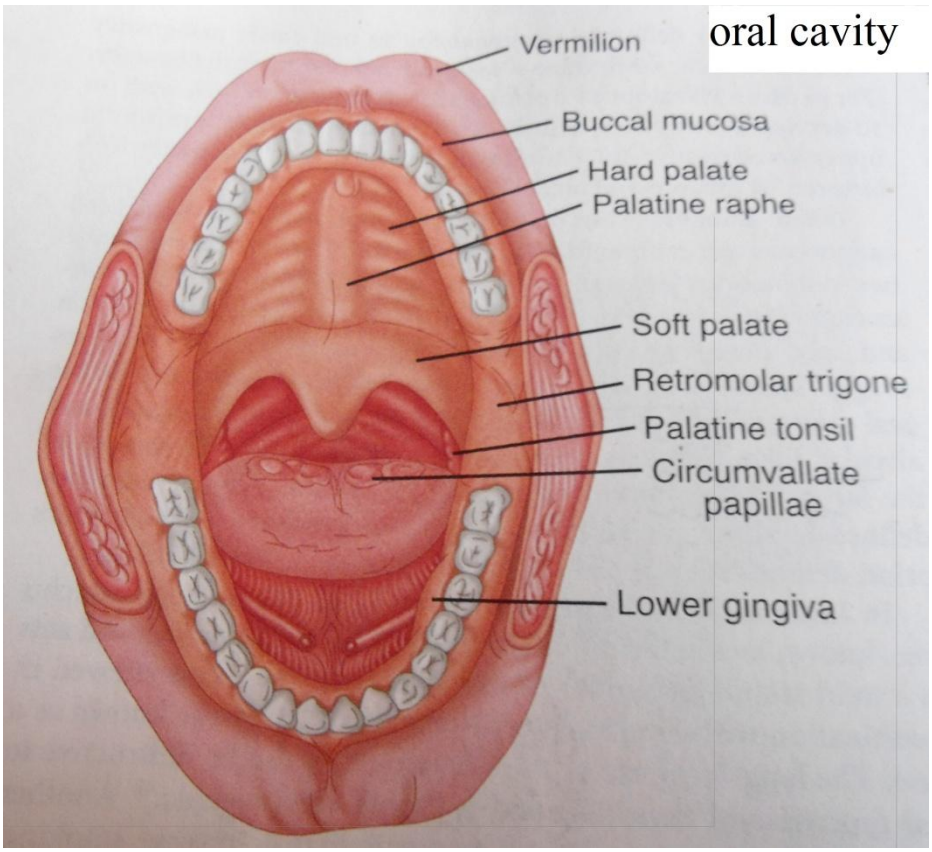
Mucosal malignant lymphoma

Secondary tumors[13]

Anatomy

The extension of oral cavity is from the vermilion border of the lip to the hard-palate/soft-palate junction superiorly, to circumvallate papillae inferiorly, and to the anterior tonsillar pillars laterally. It is further subdivided into seven sites: lips, alveolar ridges, oral tongue, retromolar trigone, floor of mouth, buccal mucosa, and hard palate.[6] With these subsites specific anatomic relationships affect diagnosis, spread, and its treatment. The contiguous spread of tumors is determined by the course of the nerves, blood vessels, lymphatic pathways, and by the fascial planes. The fascial planes serve as barriers to the direct tumor invasion and pattern of regional lymph nodal spread.[6]

Figure 1 Anatomy of Oral Cavity



Epidemiology

Male preponderance in aerodigestive tract malignancies has been noted significantly, but male-to-female ratio is steadily decreasing because of the increased incidence of female smokers. The highest incidence areas of France, Hong Kong, India, Spain, Italy, and Brazil, as well as in U.S. blacks incidence in males exceed 30 per 100,000. The highest female rate greater than 10 per 100,000 is found in association with chewing of betel quid and tobacco in India.[3] In developed countries, the incidence among the annually diagnosed neoplasms is 3% compared to Asia and India (developing countries) where it represents almost 30%.[2] In India, cancer is the second most common disease responsible for maximum mortality with about 0.3 million deaths per year.[4] Mouth and oropharynx is the most common malignancy in males

and third most common malignancy in females following cervix and breast as per ICMR 2004.[5]

Etiology

In oral mucosa, a continuous spectrum is observed beginning with preinvasive lesions, ending with invasive carcinomas and metastasis.[1] Tobacco abuse and alcoholism are the two most common risk factors which are modifiable in the development of head and neck malignancies and oral malignancies in particular.[6] The other risk factors associated are environmental UV light, poor nutrition, HPV infection, immunosuppression, chronic infection with syphilis and Plummer-Vinson syndrome.[6,14] Factors such as mechanical irritation, thermal injury, and chemical exposure have been described especially for cancer lip based on the practice of pipe smoking[6]. The risk factors are synergistic and not only additive, especially tobacco use and alcoholism.[6,15] Chronic exposure to risk factors lead to precursor/preinvasive lesions by two hit process- DNA damage and the ability of mutated cells to progress through the rest of the cell cycle, leading to invasive lesions.[6]

Carcinogenesis

Loss of cellular signaling mechanisms leads to the development of tumour. Following malignant transformation, mitosis, apoptosis and the various cellular processes of and the interaction of a cell with its surrounding environment are altered. Overexpression of mutant p53 is a key event in the malignant transformation of more than 50% of head and neck squamous cell carcinomas in smokers.

Genetic mutations may occur either de novo or due to environmental exposure to radiation or viral pathogens. Loss of heterozygosity at 3p, 4q, and 11q13 and various common mutations

in head and neck malignancies are seen more frequently in smokers.[3,6] Multiple head and neck primaries can be explained by field cancerization theory by Slaughter and associates.[6]

TNM staging of Oral cavity Carcinoma[6]	
Primary tumor	
TX	Unable to assess primary tumor
T0	No evidence of primary tumor
Tis	Carcinoma in situ
T1	Tumor is <2 cm in greatest dimension
T2	Tumor >2 cm and <4 cm in greatest dimension
T3	Tumor >4 cm in greatest dimension
T4 (lip)	Primary tumor invading cortical bone, inferior alveolar nerve, floor of mouth, or skin of face (e.g., nose or chin)
T4a (oral)	Tumor invades adjacent structures (e.g., cortical bone, into deep tongue musculature, maxillary sinus) or skin of face
T4b (oral)	Tumor invades masticator space, pterygoid plates, or skull base and/or encases the internal carotid artery
Regional lymphadenopathy	
Nx	Unable to assess regional lymph nodes
N0	No evidence of regional metastasis
N1	Metastasis in a single ipsilateral lymph node, 3 cm or less in greatest dimension

N2a	Metastasis in single ipsilateral lymph node, >3 cm and <6 cm
N2b	Metastasis in multiple ipsilateral lymph nodes, all nodes <6 cm
N2c	Metastasis in bilateral or contralateral lymph nodes, all nodes <6 cm
N3	Metastasis in a lymph node >6 cm in greatest dimension
Distant metastases	
Mx	Unable to assess for distant metastases
M0	No distant metastases
M1	Distant metastases

Group staging[6]			
Stage 0	Tis	N0	M0
Stage I	T1	N0	M0
Stage II	T2	N0	M0
Stage III	T3	N0	M0
	T1-3	N1	M0
Stage Iva	T4a	N0	M0
	T4a	N1	M0
	T1-4a	N2	M0
Stage IVb	Any T	N3	M0
	T4b	Any N	M0
Stage IVc	Any T	Any N	M1

Clinical staging is based primarily on palpation, but radiographic studies, including computed tomography (CT) and magnetic resonance imaging (MRI), have been shown to be accurate in staging the cancer as the detection of nodes clinically impalpable is amenable for study using imaging modalities.[3]

The sub classification of stage IV is as follows

Stage IVA Advanced resectable

Stage IVB Advanced unresectable

Stage IVC Advanced distant metastatic disease[3]

Grading of the primary tumour is as follows

Gx Grade cannot be assessed

G1 Well differentiated tumour

G2 Moderately differentiated tumour

G3 Poorly differentiated tumour[16]

The cervical lymphatic nodes are divided into seven levels.

1 Level I – Submental and submandibular group

- Level IA - bounded by the anterior belly of the digastric muscle, the hyoid bone, and the midline (submental triangle).
- Level IB - bounded by the anterior and posterior bellies of the digastric muscle and

the inferior border of the mandible. Level IB contains the submandibular gland along with the lymph node.

2. Level II – Upper Deep cervical group

Bounded superiorly by the skull base, anteriorly by the stylohyoid muscle, inferiorly by a horizontal plane extending posteriorly from the hyoid bone(upto hyoid bone inferiorly), and posteriorly by the posterior edge of the sternocleidomastoid muscle [between base of skull to hyoid bone].

3. Level III – Middle Deep cervical group

Begins at the inferior border of hyoid to the horizontal plane extending posteriorly from the inferior border of the cricoid cartilage. and is bounded by the laryngeal strap muscles anteriorly and by the posterior border of the sternocleidomastoid muscle posteriorly.

4. Level IV- Lower Deep cervical group

Begins at the inferior border of cricoid cartilage to the clavicle bounded anteriorly by the strap muscles and posteriorly by the posterior edge of the sternocleidomastoid muscle.

5. Level V – Posterior group

It is posterior to the posterior edge of the sternocleidomastoid muscle, anterior to the trapezius muscle, superior to the clavicle, and inferior to the base of skull.

6. Level VI – Pretracheal, paratracheal and prelaryngeal group

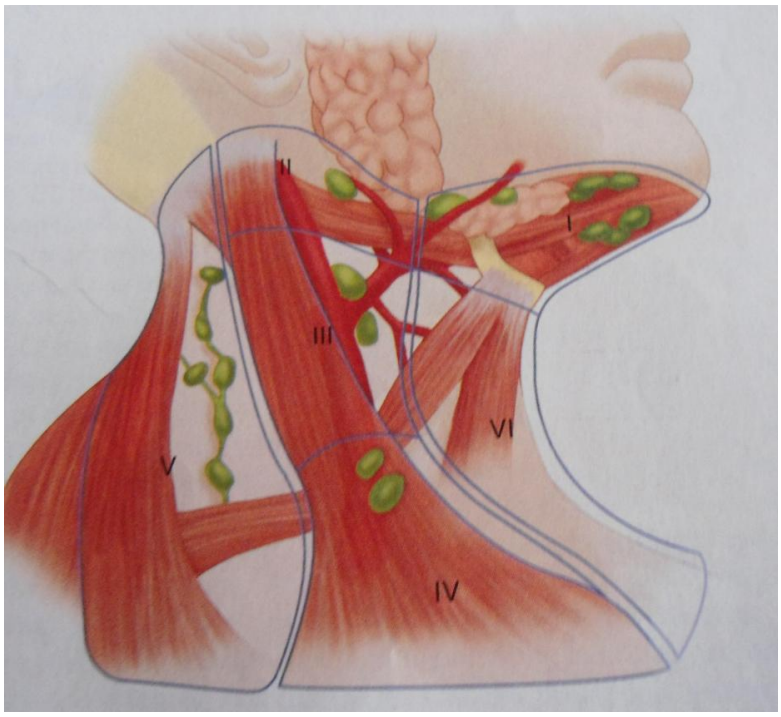


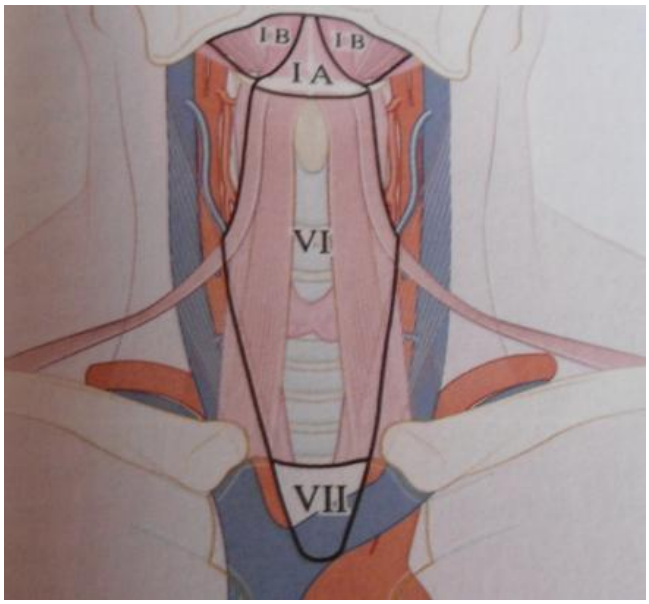
Figure 2 Levels of lymph nodes

Bounded by the hyoid bone superiorly, the common carotid arteries laterally, and the sternum inferiorly.

7. Level VII – Superior mediastinum

Lies between the common carotid arteries and is superior to the aortic arch and inferior to the upper border of the sternum.[3]

Figure 3 Levels of lymph nodes



Evaluation

Careful evaluation and accurate staging, both clinically and radiographically is required for proper treatment of oral malignancies.

Symptoms

Ulcer

Bleeding

Loose teeth

Difficulty or pain when swallowing

Difficulty wearing dentures

Lump in the neck

Earache

Pre-existing lesions – Leukoplakia, Erythroplakia, lichen planus, oral submucous fibrosis, syphilitic glossitis and sideropenic dysphagia[17]

Investigations [3]

1. Biopsy from the suspected site of malignancy
2. FNAC from cervical lymph nodes
3. Contrast CT scan of head and neck – to accurate stage the disease especially in evaluating bony destruction
4. MRI can determine soft tissue involvement excellently
5. Chest radiography or chest CT is performed to rule out synchronous lung lesions

6. Tumour markers- Alkaline phosphatase and S Calcium- not routinely done
7. Positron Emission Tomography CT – role of PET CT in head and neck cancers has not been proven comprehensively and is under investigation. The basis of PET scan is that the glucose analogue Fluorodeoxyglucose F 18 is preferentially absorbed by neoplastic cells and is evident by positron emission tomography (PET). PET has more sensitivity in identifying primary in head and neck but its sensitivity in not more than 50% in detecting unknown primary tumours.[3]

Anatomic sites

Lip

Anatomically, lip is a transition from external skin to internal mucous membrane occurring at vermilion border. Risk factors in the development of carcinoma lip are prolonged exposure to sunlight, fair complexion, immunosuppression, and tobacco use. Diagnosis of lip cancer is early as it is amenable for inspection and it bleeds on contact.

Figure 4 Lip cancer

Early lesions, T1 and T2 are usually treated by wide local excision in the form of wedge resection of the lip with primary closure. Primary radiation therapy is also highly effective

in these lesions and comparable to surgery. Larger lesions require combination therapy in the form of surgery- resection of involved tissues with neck dissections with complex reconstruction, and adjuvant radiation therapy. The size of the lesion, size of mouth and the presence of dentition determine primary closure in lip cancers after resection of primary.[18]

Buccal mucosa

Mucosa covering the cheeks and lips is called buccal mucosa. In practice, mucosa covering cheek is called buccal mucosa. It is in continuity with the gingiva of the maxillary and mandibular alveolar ridge along the buccal aspect and also retromolar trigone. Buccal SCC are commonly low grade tumours spreading submucosally along the buccinator muscle.[19] Verrucous carcinoma, a variant of SCC is seen in the buccal mucosa frequently than other regions and is indolent when compared to SCC.[15] Evaluation of a

Figure 5 Cancer Cheek with oro cutaneous fistula

buccal or gingival mucosal lesion should be done meticulously so as to address the extent of submucosal spread, osseous involvement, involvement of adjacent structures like retromolar trigone, pterygomandibular raphe, and cervical lymphatic spread which is

Figure 6 Cancer Cheek

better delineated using CT scan and MRI.[20]

Retromolar trigone

Retromolar trigone is represented by tissue posterior to the posterior inferior alveolar ridge and ascends over the inner surface of the ramus of the mandible. It is one of the areas of surgical graveyards in head and neck as the malignancy is not amenable for clinical examination easily and hence imaging evaluation of the retromolar trigone is critical. Posterior spread of cancer RMT to involve mandible is seen early.[19] It may also be involved by regional extension. CT scan seldom gives complete information because of artefacts and MRI is essential in evaluating retromolar trigone.[20]

Gingiva

Gingival/alveolar cancer tends to involve the bone in around 50% patients. The diagnosis is delayed in this region as the symptoms are neglected common dental conditions like gingivitis and periodontitis. The propensity to metastasise to lymph nodes is high but mostly confined to levels I and II. Early lesions are confined to the mucoperiosteum and resection may require a marginal mandibulectomy to remove the lesion in toto. Larger lesions involving adjacent structures like skin, floor of mouth and large areas of buccal mucosa [T4a- resectable] need extensive resection with reconstruction in the form of pectoralis major myocutaneous flap, deltopectoral flap, tongue flap, forehead flap etc.[21]

Figure 7 Cancer Gingiva

Floor of mouth

Carcinoma of floor of mouth arises within 2 cm of the anterior midline[19] and present as

painful infiltrative ulcers partly due to the involvement of the muscles of the floor of

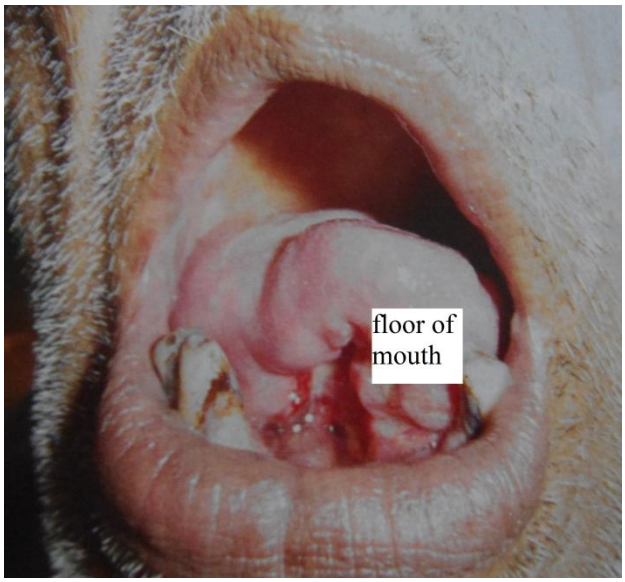


Figure 8 Cancer Floor of Mouth

mouth, and partly due to adjacent structure involvement-middle of the mandible or tongue. Submandibular and submental lymph nodes are positive for metastatic disease due to malignancy in this region. Early disease can be treated by either surgery or RT as the lesions respond to both modalities equally. Advanced lesions need surgical resection with reconstruction and adjuvant RT.[21]

Oral Tongue

Figure 9

Cancer Tongue

Incidence of cancer tongue is increasing in the present world with more and more cases diagnosed in young people. In cancer tongue, the lateral border is the most common location, followed by the anterior tongue and the dorsum.[15,19] Lymph nodal metastasis is rare in early lesions with levels II, III, I getting involved in late lesions in decreasing order. Commonest cancer in oral cavity after lip cancer and carcinoma buccal mucosa. For early disease, surgery and RT have been proven equally effective. Tumours of the Anterior third tongue invade the floor of the mouth and middle-third lesions invade the tongue musculature.[19] Excision usually entails a hemiglossectomy to obtain negative margins as tumour cells extend well beyond the clinically detected margin. In infiltrative type of cancer tongue either adjuvant RT to the neck or elective neck dissection should be considered.[21]

Hard palate

Figure 10 Cancer Hard Palate

Primary Hard palate malignancy is seen predominantly in males and is rare. Squamous cell

carcinoma comprises only 50% of the neoplasms in hard palate as minor salivary glands are seen in highest number[13]. Adenocarcinoma and adenoid cystic carcinoma is seen almost on par with squamous cell carcinoma in this region amounting to the rest. Lymph node metastasis is uncommon in hard palate (6-29%) and the presence of lymph node metastasis denotes aggressive nature of disease. Surgical treatment entails extensive resection in the form of infrastructure maxillectomy or near total palatectomy with immediate prosthetic obturator.[21]

Treatment

Treatment of oral cancer includes surgery, radiation therapy, or chemotherapy or combination therapy in the form of multimodal therapy.

Surgical- Wide local exicision with or without radical neck dissection

Radiotherapy- Curative - 50-70 Gy in 200 # for 30 cycles

Palliative tailored according to the patient[40-45Gy]

Chemotherapy- Cisplatin based chemotherapy [50- 70mg/m^2 every 3 weeks][22]

Surgery: It involves wide local excision along with a rim of normal tissue with or without lymph node dissection. Surgical procedure is based on the site of lesion, extent of lesion and presence and absence of lymphnodal involvement. Complex surgical resections are

Figure 11

Cancer Tongue- Post operative view

done in various clinical settings with flap cover to cover the resultant defect.[3,6] Time required for recovery of the patient who has undergone a complex surgical procedure is more than one who has undergone only wide local excision.[23] Also the morbidity caused by complex procedures in the form of flaps and reconstructive surgeries to be considered.

Figure 12. Cancer lip/cheek

Figure 13. Wide excision for ca lip/cheek

Figure 14. Forehead Flap

Radiotherapy: Radiation therapy uses high-energy rays to destroy cancer cells. Two types of radiation therapy to treat oral cancer:

External radiation: The radiation comes from a machine harbouring radioactive

device.

Internal radiation (implant radiation): Radiation comes from radioactive material placed in seeds, needles, or thin plastic tubes placed directly in the tissue. The implants remain in place for several days.

Both internal and external devices may be used for the same patient.[23]

Radiation doses are chosen as to maximize tumor control with acceptable side effects. Complete response is when the size of tumor reduces from 3 cm diameter to 3 mm, which still leaves 100 million tumor cells as normally a tumor of size one cm cube contains one billion cells. Each radiation fraction kills a certain amount of tumor fraction, the radiation dose required to cure occult cancer is more. With standard fractionation, 45 – 54 Gy is used when there is moderate suspicion of occult disease as adjuvant radiotherapy, 60 – 65 Gy for high suspicion of occult disease or for positive margins and for grossly left over tumor, the dose is more than 70 Gy.[22]

The standard fractionation of RT is defined as the delivery of one treatment of 1.8 to 2.25 Gy per day. This is based on the understanding of chance of tumor control and risk of normal tissue damage. Accelerated fractionation is a technique in RT which has developed from head and neck malignancies due to the fact that protracted treatment due to increasing radiation dose was associated with loss of tumour control due to accelerated repopulation. Accelerated fractionation completes RT before tumour cells enter the accelerated repopulation phase, in which after administering the standard radiation dose in the morning, concomitant boost [second treatment to boost field in the afternoon] is given. Hyperfractionation uses the same principle of accelerated fractionation, but the dose per fraction is less than the standard fraction with more fractions delivered to the patient. This is expected to cause less late complications for the same acute effects on tissues, both

normal and tumour.[22]

Hypofractionation is the technique of administration of smaller number of fractions of higher dosages, used in the setting of palliative RT and is known to cause more late toxicity. [22]

Shrinking field technique is a technique used in the setting of gross disease, where 50 Gy dose is delivered to the entire region and additional boost dose of 20 Gy to the tumor. The principle behind this is that gross tumor invariably resides within the region at risk for radiotherapy. With the development of intensity modulated RT [IMRT], both the regions are treated simultaneously achieving the goals. [22]

RT alone is used as curative modality in the setting of localized tumors. The decision to go for RT or surgery is determined by tumor characteristics. If the risk of recurrence after surgery is low, or if the recurrence is easily controlled by second resection, surgery is preferred. Whereas if the morbidity is more with tumor resection, gross tumour resection is still associated with high risk of residual disease, RT is preferred. Adjuvant RT can reduce local failure rates to below 10% if gross tumor resection is achieved. [22]

In neoadjuvant setting, RT is preferred in head and neck cancers as this setting can be readily administered than adjuvant therapy with less morbidity than with adjuvant setting and efficacy almost equal to adjuvant RT.

Indications for RT in adjuvant setting[22] are

Close [5 mm] or positive margin

Extracapsular extension

Multiple positive nodes

Invasion of soft tissues of neck

Endothelial-lined space invasion

Perineural invasion

Long term survival benefit from RT in malignancies is achieved by the use of effective chemotherapy along with RT by preventing deaths due to early metastatic disease especially if CT is moderately effective. Adjuvant radiation has little influence on survival benefit if CT is ineffective or very effective, as the systemic relapse dominates local tumor control by RT and survival benefit is not noted.

Palliative RT in contrast to radical treatment should weigh the anticipated toxicity against potential benefit in treating the patient whereas radical treatment aims in eradicating the disease in spite of treatment associated morbidity.

The side effects of radiation therapy depend mainly on the radiation dose given.

- Dry mouth
- Tooth decay
- Sore throat or mouth
- Sore or bleeding gums
- Infection
- Delayed healing after dental care
- Jaw stiffness
- Denture problems
- Changes in the sense of taste and smell

- Changes in voice quality
- Changes in the thyroid
- Skin changes in the treated area
- Fatigue[23]

Chemotherapy: Chemotherapy uses anticancer drugs to kill cancer cells. It is called systemic therapy because it enters the bloodstream and can affect cancer cells throughout the body.[23] In head and neck cancers, neoadjuvant chemotherapy is indicated in the setting of a locally advanced disease and there is an expanding role of primary chemotherapy in advanced disease. The primary indication of CT in head and neck cancers are metastatic disease and in locally advanced disease, it is used as induction therapy to help to reduce tumour burden and to proceed with surgery.[22] In early disease CT has no much role as the local tumour burden is amenable either to surgery or RT.

Initially single agent CT was used for head and neck cancers. Various chemotherapeutical agents were used in this setting and their efficacy tested. But single agent CT did not show any significant increase in survival, including taxanes. The most commonly used drugs were methotrexate and cisplatin. The various drugs and their dosages in single agent CT are as follows[22]

Inj Methotrexate 40 -60 mg/m^2 intravenously once every week

Inj Cisplatin 75- 100 mg/m^2 intravenously once every 3 -4 weeks

Inj Paclitaxel 135 – 225 mg/m^2 intravenously for 3 hours once every 3 weeks

Inj Docitaxel 60 -100 mg/m^2 intravenously for 3 hours once every 3 weeks

The various other drugs used as single agent CT are Carboplatin, 5 Flurouracil, EGFR inhibitor Cetuximab and tyrosine kinase inhibitors Erlotinib and Geftinib.

Since the role of single agent CT was disappointing in disease control and improving survival, combination chemotherapy came in to practice. Most of combination CT regimens consider Cisplatin to which are added other chemotherapeutic agents. Combination CT has shown improvement in the form of improved survival when compared to single agent CT both statistically and clinically and has been confirmed by the ECOG group [Eastern Co operative Oncology Group]. [22] CT being a systemic form of treatment has various side effects, some related to systemic treatment and others particular to each chemotherapeutic agent. And side effects are common with combination chemotherapy when compared to single agent CT.

Combination CT of Docetaxel and Cisplatin has shown response rate of 40 – 53% with CR of 16 – 18%. Cisplatin and 5 FU combination has also showed similar response rate. The addition of Interferon alpha 2b to the above combination did not improve the survival rate.

Paclitaxel in the dose of 80 mg/m^2 with Carboplatin 175 – 200 mg/m^2 area under curve 2 [AUC 2] over 3 hours with Carboplatin AUC 6 has improved survival rate in advanced head and neck cancers.The study of single agent Cisplatin Vs Cisplatin along with Cetuximab has shown definite improvement in overall survival.

The combination of CT and RT in the form of concurrent CT and RT has improved 2 year survival rate to more than 20%. Sequential RTOG studies has proved the same and the most commonly used regimen is

Paclitaxel 20 mg/m2 intravenously + Cisplatin 15 mg/m2 intravenously + RT 60 Gy with

1.5 Gy fractionation twice daily along with Granulocyte Colony Stimulating Factor. [22]

Determination of response to Chemotherapy

WHO Change in Sum of Products [22]

Complete response[CR] – Disappearance of all target lesions without any residual lesion confirmed at 4 weeks

Partial response[PR] – 50% or more reduction in size target lesions without a 25% increase in any one of target lesion confirmed at 4 weeks

Stable disease – When neither PR or progressive disease [PD] criteria are met

Progressive disease[PD] – 25% or more increase in size of measurable lesion or appearance of a lesion

RECIST Change in Sum Longest Diameter [22]

CR – disappearance of all target lesions confirmed at 4 weeks

PR – At least 30% reduction in the sum of longest diameter of target lesions confirmed at 4 weeks taking baseline study as reference

Stable disease - When neither PR or progressive disease [PD] criteria are met, taking as reference the smallest sum of the longest diameter recorded since treatment started

PD - At least 20% increase in the sum of longest diameter of target lesions, taking as reference the smallest sum of the longest diameter recorded since treatment started or appearance of new lesions

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

1. To study the common sites of involvement in various subgroups
2. To correlate FNAC and biopsy results in oral cavity lesions
3. To study the various clinical and histopathological factors implicated in prognostication of oral malignancies
4. To study the impact of histopathological grading on treatment and predictors of advanced disease
5. To evaluate the response to treatment of oral malignancy with special reference to radiotherapy and chemotherapy
6. To compare early and late oral malignancies and their response to treatment
7. To study oral cavity malignancy with special interest towards synchronous and metachronous head and neck malignancies

MATERIALS AND METHODS

MATERIALS AND METHODS

Patients of oral malignancy admitted in TvMCH during the period from February, 2011 to August, 2012 has been considered as the study group, as per the inclusion and exclusion criteria given below

Inclusion criteria: Age > 18 years

Previously diagnosed as a case of oral malignancy and on treatment

Patients with synchronous and metachronous head and neck malignancies

Exclusion criteria: Age < 18 years

Known serious medical comorbidities

Pregnancy

Major surgery within 14 days prior

Design of study: Prospective analysis of case series

Sample size: 98

Patients were evaluated based on questionnaires and the records were scrutinized for relevant clinical, pathological and treatment protocols.

The various indicators which are under study are: age, sex, demographic data, risk factors, immune status, macroscopic appearance, size of lesion, surrounding induration, bony involvement, depth of invasion, regional lymph nodal involvement.

A thorough history, clinical examination and investigations are carried out and patients are staged, treated and followed up during the study period. Histopathological grading and

adequate resection margins are taken into consideration. Operative morbidity and mortality, various chemotherapeutic agents, radiation and their serious adverse effects, if any, are recorded.

Samples (FNAC, biopsy- excision and wedge) are taken and are sent to pathology department for histopathology.

Treatment protocol in TvMCH

Surgical- Wide local excision with or without neck dissections

Wide local excision with reconstruction using flaps

Radiotherapy- Curative - 50-70 Gy in 200 # for 30 cycles

Palliative tailored according to the patient [40-45Gy]

Chemotherapy- Cisplatin based chemotherapy [50- 70mg/m^2 every 3 weeks]

Outcome measures

The outcome is being evaluated with the following parameters

- Locoregional control
- Development of second malignant neoplasm/ recurrence

Statistical analysis

The statistical analysis of the data is being done by using measures of central tendency (mean, median, mode) Chi-square test and data packages in case, if required.

National significance

Malignancies of head and neck, especially that of oral cavity is common in the Indian sub-continent which is in advanced stage at the time of diagnosis with a poor 5 year survival rate. Prognostication of factors in malignancies helps in systematic approach and sheds light towards grey areas.

RESULTS

RESULTS

Table 1 Age distribution

Age(yrs)	30-39	40-49	50-59	60-69	70-79	80-89	total
Study	6	15	27	29	20	1	98
L. P. Dragomir et al[1]	4	13	31	45	22	2	117

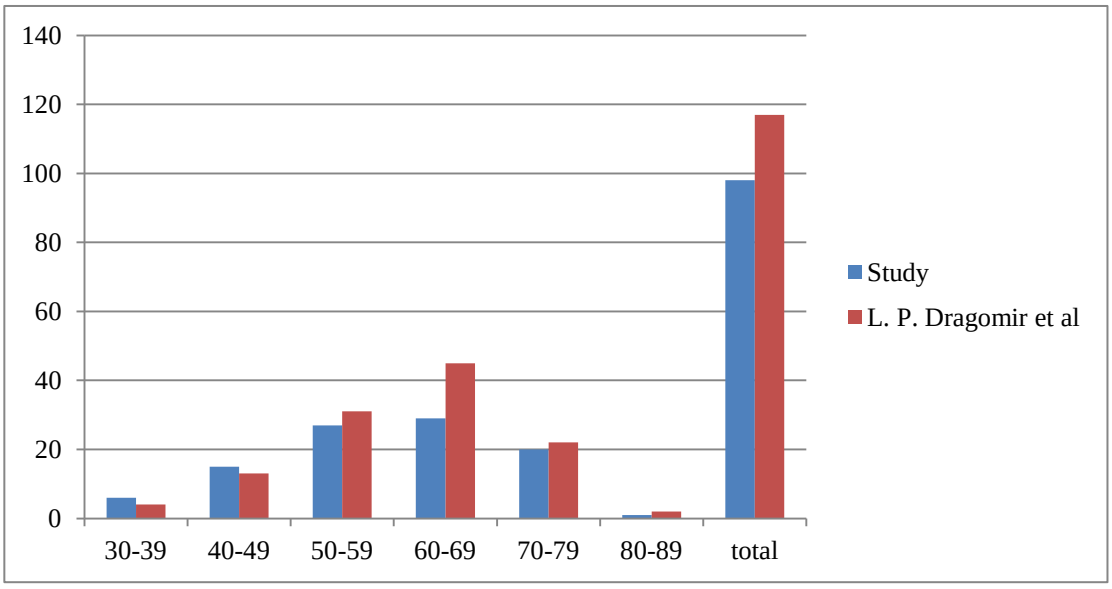


Figure 15 Age distribution

The maximum number of patients belonged to the age group of 60- 69 years in both study group and L.P. Dragomir et al. Most of the patients fall in the age group of 50 – 79 years which is 76 in study group and 98 in L.P. Dragomir et al.

Table 2 Macroscopic type

Type	Ulcerative	Ulceroproliferative	Proliferative	Infiltrative	
Study	43	36	4	15	98
L. P. Dragomir et al[1]	48	24	31	14	117

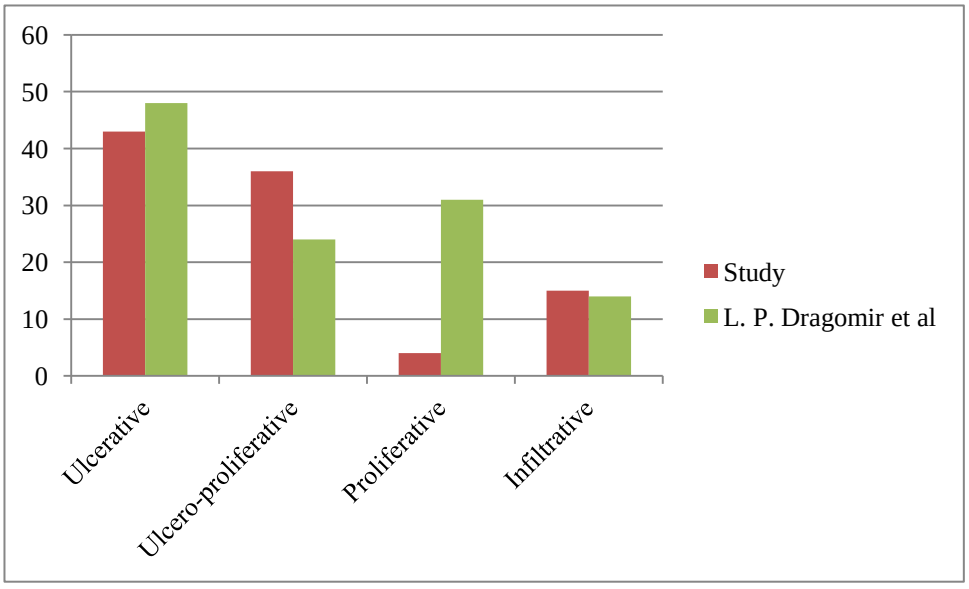


Figure 16 Macroscopic type

In morphology of the lesion, ulcerative was the commonest variety [43%] found in both the study group and L. P. Dragomir et al [41.02], which was followed by ulceroproliferative type [37%].

Table 3 Dimensions of lesion

Size(cm)	< 2	2-4	>4(T3)	>4(T4)	
Study	24	71	0	3	98
L. P. Dragomir et al[1]	11	31	15	7	64

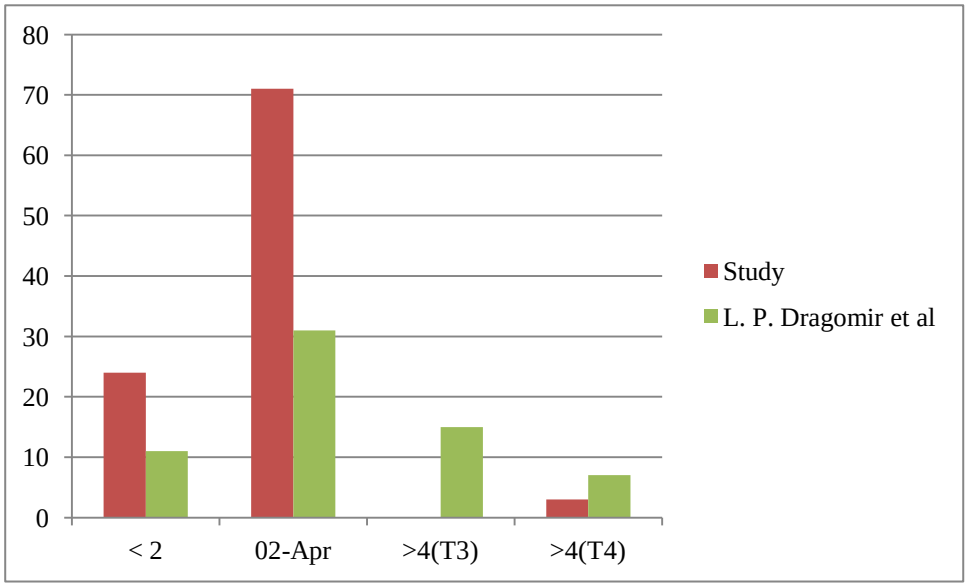


Figure 17. Dimensions of lesion

T2 lesions were found to be predominating in both the study group [73%] and L. P. Dragomir et al [48%], followed by T1 lesions [24%].

Table 4 Sex distribution

Sex	Male	Female	
Study	72	26	98
L. P. Dragomir et al[1]	105	12	117

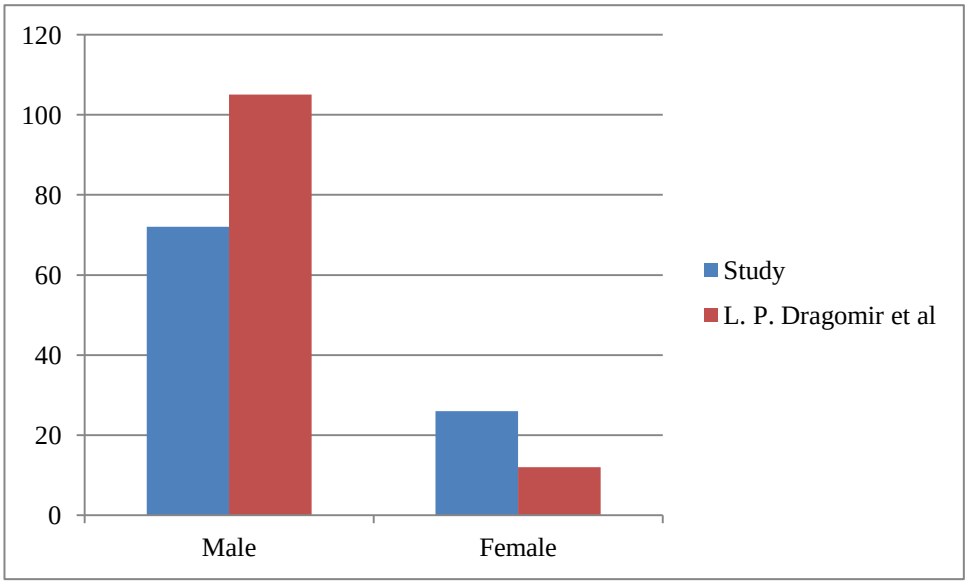


Figure 18 Sex distribution

Gender is not a risk factor in the development of oral malignancies, but is commonly seen due to the fact that risk factors in oral malignancy are seen more in males. Males constituted about 70% of study and 89% of L. P. Dragomir et al group.

Table 5 Risk factors

Risk factors	Smoking	Alcoholism	Pre-existing lesions	Tobacco chewing	STD	FH	Associations
Study	56	24	86	49	-	-	92
L. P. Dragomir et al[1]	11	7	5	-	-	1	21

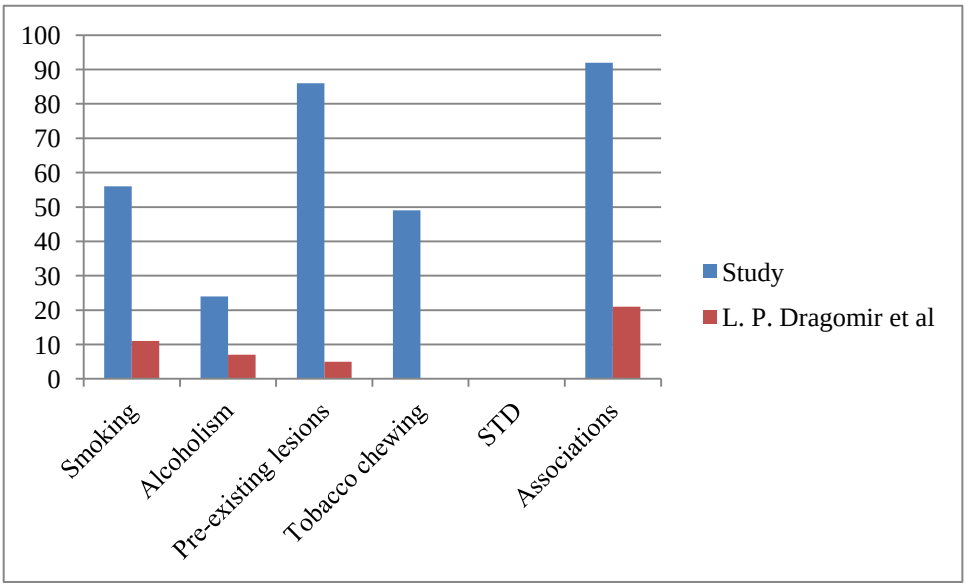


Figure 19 Risk factors

Smoking was seen in 57% of study group, while other associated risk factors tobacco chewing and alcoholism were seen in 50% and 24% of study group respectively. The association between various risk factors was up to 92%.

Table 6 Localisation of lesion

Localisation	Lip			Cheek		Tongue		Gingiva		RMT		Hard palate	Floor of mouth	
	UL	LL	Angle	Rt	Lt	Rt	Lt	Rt	Lt	Rt	Lt			
Study	2	1	2	22	14	17	18	1	2	1	3	11	4	98
	5			36		35		3		4		11	4	
L. P. Dragomir et al[1]	41			-		38		26		-		5	7	117

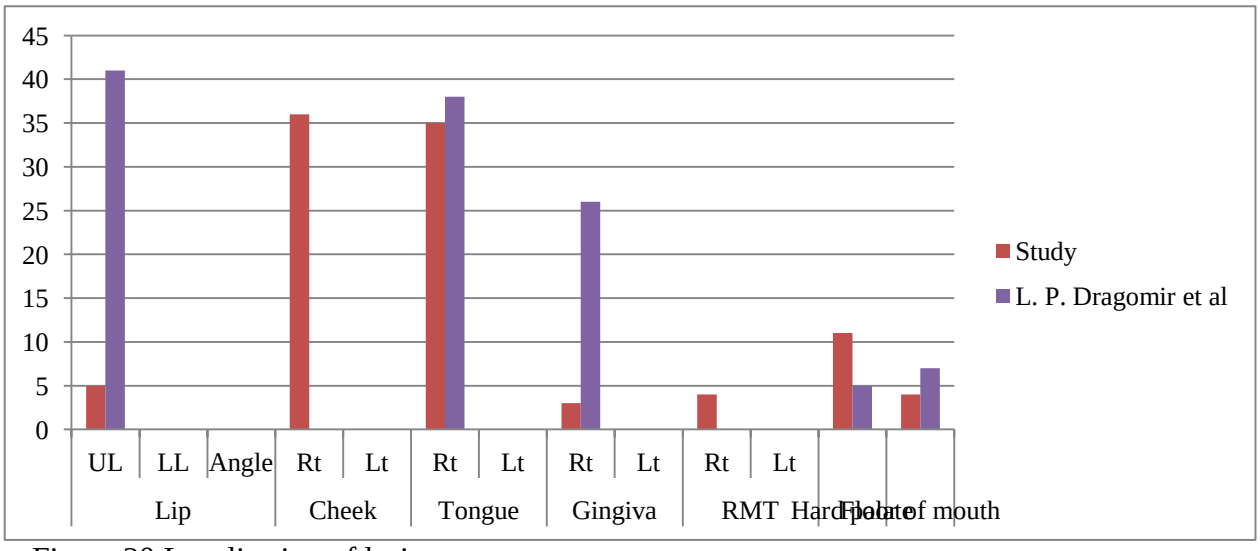


Figure 20 Localisation of lesion

Cancer buccal mucosa and tongue were the commonest types accounting to 37% and 36% respectively in the study group comprising 71% of overall oral cancers. Whereas in L. P. Dragomir et al group, cancer lip [35.04%] was followed by tongue [32%]. This is probably explained by the fact that in the Indian scenario, tobacco chewing is more common when compared to Western World.

Table 7 Differentiation

Differentiation	Well differentiated	Moderately differentiated	Poorly differentiated	
Study	45	27	26	98
L. P. Dragomir et al[1]	44	32	42	118

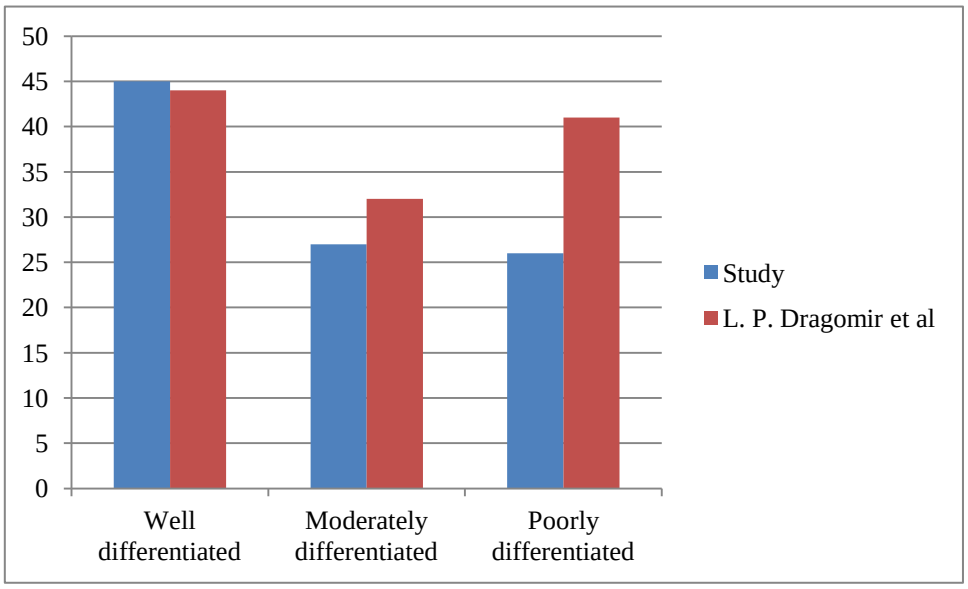


Figure 21 Differentiation

In both study and L. P. Dragomir et al groups, well differentiated tumours were common to the tune of 45% and 37% respectively.

Table 8 Tumor progression

Progression	Microinvasive carcinoma	Invasive carcinoma without nodal metastasis	Invasive carcinoma with nodal metastasis	
Study	-	19	79	98
L. P. Dragomir et al[1]	2	57	3	62

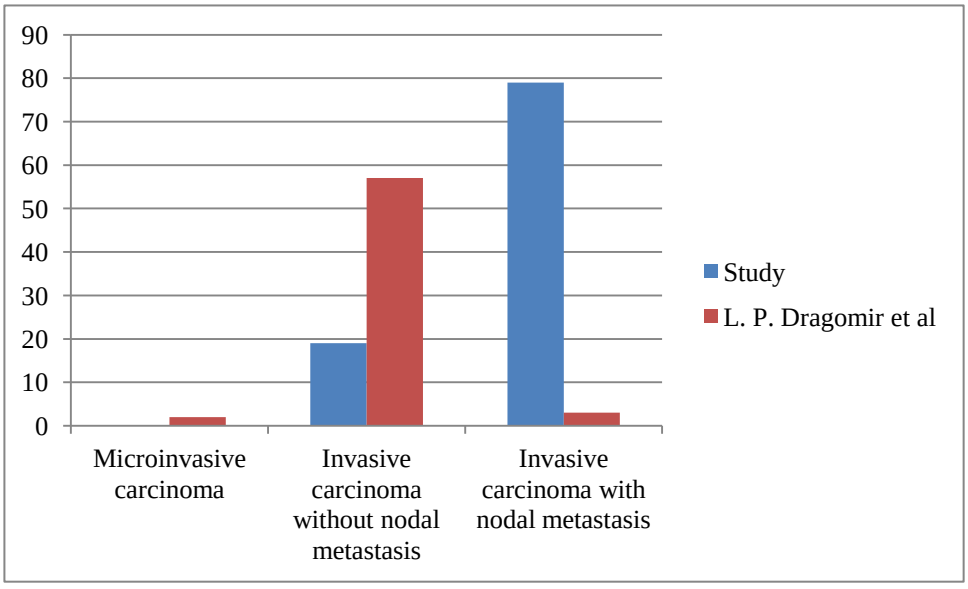


Figure 22 Tumor progression

Invasive cancer with nodal metastasis was common in the study group [80%], probably owing to the late presentation of cancers.

Table 9 Treatment

Treatment	Radiotherapy	Surgery(S)	Chemotherapy	Combined	Symptomatic
	78	5	10	4 S + RT 3 CT + RT 1	1
	79%	5%	10%	4%	1%

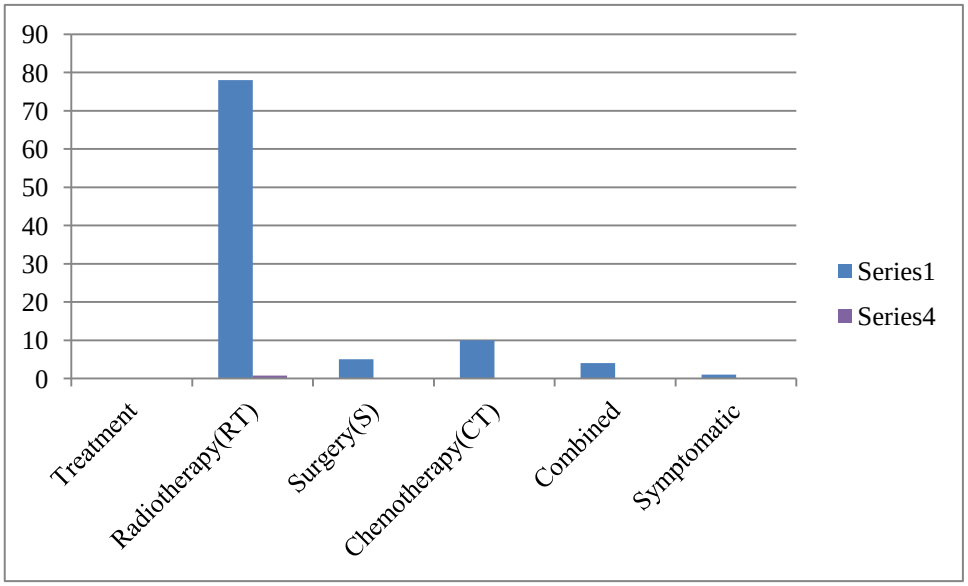


Figure 23 Treatment

Most of the cases are treated in TvMCH by RT. Hence the study group has received RT to the tune of 80%.

Table 10 Resected margins (Total no of surgical cases 8)

Margins	Free of tumor	Involved by tumor	
Study	5	3	8
L. P. Dragomir et al[1]	12	13	25

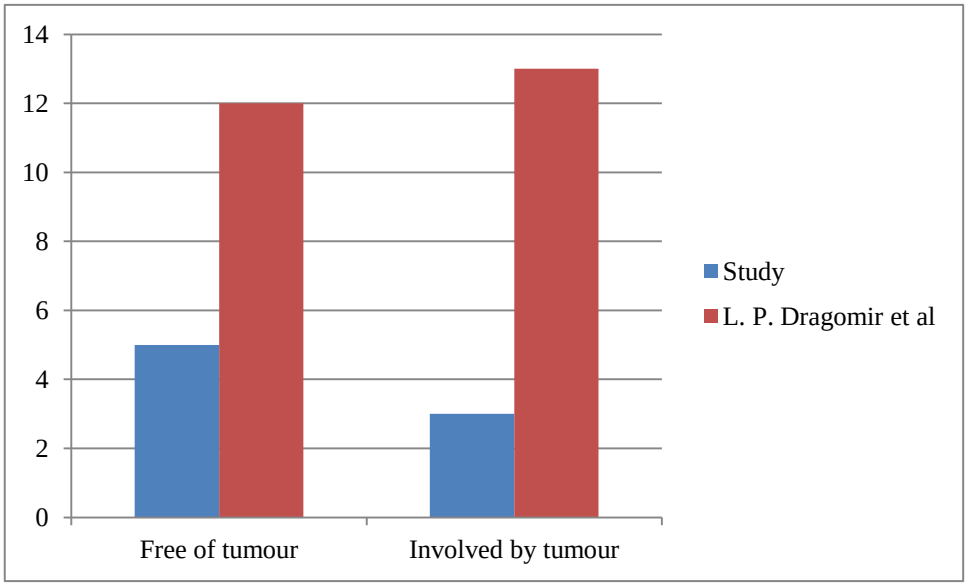


Figure 24 Resection margins

Of the eight patients taken up for surgery, five had margins free of tumour.

Individual statistics

Table 11 Age distribution - lesion wise- Most of cases belonged to 50 – 79 years with the exception of tongue where maximum cases were in 40 – 49 year age group.

Age(yrs)	30 - 39	40 -49	50 -59	60 -69	70 – 79	80 - 89	No
Lip	-	1	2	2	-	-	5
Cheek	3	3	8	12	10	-	36
Tongue	2	10	9	8	6	-	35
Hard palate	1	1	3	4	2	-	11
RMT	-	-	2	1	1	-	4
Gingiva	-	-	1	-	1	1	3
Floor of mouth	-	-	3	1	-	-	4

Table 12 Macroscopic type – lesion wise- Most of cheek lesions were ulceroproliferative, tongue lesions were ulcerative and hard palate infiltrative.

Type	Ulcerative	Ulceroproliferative	Proliferative	Infilterative	No
Lip	2	2	1	-	5
Cheek	1	31	3	1	36
Tongue	29	2	1	3	35
Hard palate	2	1	-	8	11
RMT	1	-	-	3	4
Gingiva	1	2	-	-	3
Floor of mouth	-	1	-	3	4

Table 13 Dimensions - lesion wise- Most cases belonged to T2 group [2-4 cm].

Dimensions	< 2 cm	2 – 4 cm	T3	T4	No
Lip	5	-	-	-	5
Cheek	2	30	1	3	36
Tongue	9	26	-	-	35
Hard palate	5	6	-	-	11
RMT	1	3	-	-	4
Gingiva	1	2	-	-	3
Floor of mouth	1	3	-	-	4

Table 14 Sex distribution - lesion wise- Sex predilection was seen for Cancer tongue and Cancer hard palate.

Sex	Male	Female	No
Lip	3	2	5
Cheek	20	16	36
Tongue	31	4	35
Hard palate	8	3	11
RMT	3	1	4
Gingiva	3	-	3

Floor of mouth	4	-	4

Table 15 Risk factors – lesion wise- Main risk factors were Smoking and tobacco chewing.

Risk factors	Smoking	Alcoholism	Lesions	Tobacco chewing	STD	Associations
Lip	3	3	4	2	-	1
Cheek	18	6	35	20	-	36
Tongue	21	10	30	13	-	35
Hard palate	6	1	6	7	-	7
RMT	3	2	3	2	-	4
Gingiva	3	1	3	1	-	3
Floor of mouth	4	1	3	-	-	4

Table 16 Differentiation - lesion wise- Most cases were well differentiated but for retromolar trigone where poorly differentiated variety was seen commonly.

Differentiation	Well	Moderate	Poor	No
Lip	4	-	1	5
Cheek	19	9	8	36
Tongue	16	11	8	35
Hard palate	3	3	5	11

RMT	-	1	3	4
Gingiva	1	-	2	3
Floor of mouth	2	2	-	4

Table 17 Tumour Progression - lesion wise- Most cases had nodal metastasis.

Progression	Invasive carcinoma with no nodal metastasis	Invasive carcinoma with nodal metastasis	No
Lip	1	4	5
Cheek	1	35	36
Tongue	3	32	35
Hard palate	11	-	11
RMT	1	3	4
Gingiva	1	2	3
Floor of mouth	1	3	4

Table 18 Treatment - lesion wise

Treatment	RT	Surgery(S)	CT	Combined	Symptomatic	No
Lip	3	1	-	-	1	5
Cheek	29	4	2	1 S + RT	-	36

Tongue	28	-	5	2 S + RT CT + RT	-	35
Hard palate	9	-	2	-	-	11
RMT	4	-	-	-	-	4
Gingiva	3	-	-	-	-	3
Floor of mouth	2	-	1	1 S + RT	-	4

Table 19 Resected margins (surgical cases 8) - lesion wise- Cancer cheek had high tumor free margins.

Treatment	Free of tumor	Involved by tumor	Percentage Of surgical cases
Lip	1	-	20%
Cheek	4	1	13%
Tongue	-	1	2%
Hard palate	-	-	0%
RMT	-	-	0%
Gingiva	-	-	0%
Floor of mouth	-	1	25%

Table 20 Staging – lesion wise

Treatment	I	II	III	IV	No
Lip	1	-	4	-	5
Cheek	1	-	8	27	36
Tongue	3	-	17	15	35
Hard palate	5	6	-	-	11
RMT	-	1	3	-	4
Gingiva	-	1	1	1	3
Floor of mouth	1	-	2	1	4

Table 21 History

	Pre existing Lesion	Toothloss	Swallowing Difficulty	Salivation	Bleeding	Difficulty-mouth opening	Weight Loss	Smoking	Tobacco Chewing	Alcohol
Lip	4	-	-	1	2	2	-	3	3	2
Cheek	35	6	12	14	17	15	2	18	20	6
Tongue	30	5	13	24	15	4	-	21	13	10
Hard palate	6	-	-	3	1	-	-	6	7	1
RMT	3	-	4	4	-	-	-	3	2	2
Gingiva	3	3	-	-	3	-	-	3	1	1
Floor of mouth	3	1	2	3	1	-	-	4	1	-

Subsites in oral cavity

Table 22 Lip (Cases: 5) – Early lesions had CR when compared to late lesions.

No of cases	Stage	Response			Smoking	Alcoholism	Differentiated			Tobacco
		Complete	Partial	No Response			Well	Mod	Poor	
1	I	1	-	-	1	1	1	-	-	-
-	II	-	-	-	-	-	-	-	-	-
4	III	1	2	1	2	2	3	-	1	-
-	IV	-	-	-	-	-	-	-	-	-

Table 23 Cheek (Cases: 36) – More lesions were late lesions and the response was partial.

No of cases	Stage	Response			Smoking	Alcoholism	Differentiated			Tobacco
		Complete	Partial	Recurrence			Well	Mod	Poor	
1	I	1	-	-	1	-	1	-	-	1
-	II	-	-	-	-	-	-	-	-	-
8	III	2	6	-	5	1	5	2	1	5
27	IV	2	24	1	12	3	13	7	7	18

Table 24 Tongue (Cases: 35) - More lesions were late lesions and the response was partial.

No of Cases	Stage	Response		Smoking	Alcoholism	Differentiated			Tobacco
		Complete	Partial			Well	Mod	Poor	
3	I	2	1	2	-	3	-	-	3
-	II	-	-	-	-	-	-	-	-
17	III	2	15	11	7	7	6	4	6
15	IV	-	15	13	4	6	5	4	5

Table 25 Hard palate (Cases: 11) – Most of the lesions were early and the response being good.

No of Cases	Stage	Response		Smoking	Alcoholism	Differentiation			Tobacco
		Complete	Partial			Well	Mod	Poor	
5	I	4	1	4	1	3	-	2	3
6	II	6	-	4	-	-	3	3	3
-	III	-	-	-	-	-	-	-	-
-	IV	-	-	-	-	-	-	-	-

Table 26 Retromolar trigone (Cases: 4) – Most cases were poorly differentiated and response being partial.

No of Cases	Stage	Response		Smoking	Alcoholism	Differentiation			Tobacco
		Complete	Partial			Well	Mod	Poor	
-	I	-	-	-	-	-	-	-	-
1	II	-	1	1	-	-	1	-	1
3	III	-	3	2	2	-	-	3	1
-	IV	-	-	-	-	-	-	-	-

Table 27 Gingiva (Cases: 3) – Late lesions had Partial Response.

No of Cases	Stage	Response		Smoking	Alcoholism	Differentiation			Tobacco
		Complete	Partial			Well	Mod	Poor	
-	I	-	-	-	-	-	-	-	-
1	II	1	-	1	1	-	-	1	-
1	III	-	1	1	-	1	-	-	1
1	IV	-	1	1	-	-	-	1	-

Table 28 Floor of mouth (Cases: 4) – Late lesions had less favorable outcome.

No of Cases	Stage	Response		Smoking	Alcoholism	Differentiation			Tobacco
		Complete	Partial			Well	Mod	Poor	
1	I	1	-	1	1	-	1	-	-
-	II	-	-	-	-	-	-	-	-
2	III	1	1	2	-	2	-	-	-
1	IV	-	1	1	1	-	1	-	-

Table 9 TREATMENT

Treatment	Radiotherapy	Surgery(S)	Chemotherapy(CT)	Combined	Symptomatic
No	78	5	10	4 S + RT 3 CT + RT 1	1

Chemotherapy : **10 cases**

Two cases of Ca cheek – stage IV

Five cases of Ca tongue – four stage III

one stage IV

Two cases of Hard palate

One case of Floor of mouth

Combined treatment modality - **Four**

Surgery + Radiotherapy - **Three** cases -Ca tongue (stage I)

Ca cheek (stage IV)

Ca floor of mouth (stage III)

Radiotherapy + Chemotherapy- **One** case of Ca tongue (stage IV)

Surgery - **Eight**

Ca tongue **One** stage I

Ca lip **One** stage I

Ca cheek **Four** - 3 cases of stage III and one stage IV

Ca cheek/lip **One** stage IV

Ca Floor of mouth **One** stage III

Recurrence

One case of Ca cheek stage IV After one month of surgery + RT

Synchronous malignancy

One case of Ca lip/cheek Stage IV

Table 29 **TREATMENT OUTCOME**

OUTCOME	NUMBER OF CASES	PERCENT
Complete relapse	24	24%
Partial relapse	72	73%
No response	1	1%
Recurrence	1	1%
Synchronous malignancy	1	1%

Complete relapse was seen in 24 cases and partial relapse in 72 cases.

DISCUSSION

DISSCUSSION

Localization of lesion

Oral malignancies after diagnosis is broadly categorized based on the anatomical location. Of the 98 patients taken up under the study, the majority belonged to Carcinoma buccal mucosa [36 patients-36%] and carcinoma tongue [35 patients- 35%] making up to 72% of the study group. Carcinoma alveolus/gingiva forms the lowest cohort [3 patients- 3%].

In contrast in L. P. Dragomir et al[1] group, cancer lip [41 patients-35.04%] and tongue [38 patients-32.47%] had higher incidence compared to other regions with an overall percentage of 67.52% with hard palate forming lowest cohort [5 patients- 4.2%].

The difference can probably be explained by the high incidence of betal nut [tobacco] chewing in India and increased exposure to sun light in the reference group [L. P. Dragomir et al[1]].

Patel MM and Pandya AN[24] studying oral and oropharyngeal cancers have reported the commonest site as oral tongue [23.02%], followed by posterior third tongue [19.64%], alveolus, lips and cheek.

Mehrotra Ravi et al,[25] in their study, cancer tongue was the most common sub site with 129 cases [42.57%] of followed by the cheek in 58 [19.14%].

Iype EM et al,[26] has reported that the commonest site in oral cancer is tongue cancer at 52% followed by buccal mucosa [26%], alveolus [10%], palate [4.5%], lip [2.3%] and floor of mouth forming the least common variety [1.9%].

Khandekar SP et al,[27] have showed alveolus as the commonest site of presentation with 55% of cases. In Ahluwalia et al[28] in the year 2001, carcinoma cheek was the common oral malignancy at 55.60% followed by oral tongue at 31.41%.

The study shows similar distribution of cancer in various sites when compared to Indian studies. Cancer cheek and tongue cancer have almost similar presentation rates, 36% and 35% respectively.

Sex

The sex ratio in the study group and L. P. Dragomir et al[1] group has clearly demonstrated the male preponderance in oral malignacies and head and neck cancers in general with male preponderance being 73% and 89.74% respectively.

Table 30 Sex distribution in various studies

Study	Male [%]	Female [%]
Patel MM et al[24]	75	25
Mehrotra et al[25]	76.57	23.43
Iype et al[26]	70	30
Dhar PK et al[29]	68.3	31.7
Khandekar et al[27]	61.25	38.75

Sex is not a risk factor for the development of oral cancers in males[32]. The difference in gender is due to high rate of smoking and alcoholism in males. Also tobacco chewing is also high among men in the Indian sub continent. The study has shown the male population is more affected similar to various studies reported in India as mentioned in the table.

Age

The age group between 50-79 years had the highest incidence of cases [76 patients] with a percentage of 77% which is similar to the study conducted by L. P. Dragomir et al[1] with the incidence of 98 out of 117 cases in similar age group [83.76%].

The youngest age of presentation in the study group is 30 year old female with carcinoma oral tongue stage III and the oldest age was 82 year old male with stage III cancer gingiva. Only six percent were less than 40 years belonging to the age group 30 – 39 years.

The maximum number of patients belonged to the age group of 60- 69 years in both study group [29 cases- 29%] and L.P. Dragomir et al group[1] [38.46%], which was followed by 27 cases in the age group 50 – 59 years and 20 cases in 70 – 79 year group.

Patel MM et al[24] have reported in his study on oral and oropharyngeal cancers, 12.9% of cancers below 35 years, 23.8% of cases between 35 – 45, and 63.3% cases over the age of 45 years.

Mehrotra Ravi et al,[25] in a study on oral cancer, has reported that the maximum incidence was in the age group 50 – 59 years. Iype et al has reported an incidence of 2.8% of cases before the age of 35years. Dhar PK et al[29] reported maximum incidence [35.7%] in the age group of 51- 60 years.

Since most of the studies show that that the incidence of oral cancer is more after the age of 50 years, targeting screening programmes to males over 50 years with smoking can detect cancers at an early stage.

Morphology of lesion

In gross morphology, the study group had more number of ulcerative [43%] and ulceroproliferative [36%] types when compared to infiltrative [15%] and proliferative [4%] types making up to 80% of lesions. Whereas in L. P. Dragomir et al group[1], due to more proliferative [26.49%] type lesions, ulcerative [41.02%] and ulceroprolifertive [20.51%] types constituted to about 61.53%.

The difference is due to high incidence of cancer lip in the reference population, which has proliferation as the common morphological pattern.

Size of lesion

With respect to the size of the lesion, T2 lesions[2-4 cm] constituted the largest group in both study and L. P. Dragomir et al[1] group making 72% and 48% respectively followed by T1 lesion[less than 2 cm] in study and T3 lesion[more than 4 cm resectable lesion] in L. P. Dragomir et al group.[1]

Size of the lesion determines treatment outcome and also the choice of surgery.[31] Only tumour depth and grade is associated with lymphnodal spread. Tumor size [diameter] and margins predicts local recurrence.[30]

Hence the size as well as the depth of the lesion helps in prognosticating the disease.[30]

Tumour Type and Differentiation

All cases in the study group were found to be of squamous cell origin similar to various studies conducted on oral cancers.

Table 31 Tumour Type in various studies

Study	Percentage of SCC[%]
Patel MM et al[24]	100
Mehrotra et al[25]	100*
Alhuwalia et al[28]	92.56
Iype EM et al[26]	72
Khandekar et al[27]	72.5

*all the malignant cases confirmed by biopsy were SCC with varying grades. Benign & premalignant lesions were also part of study.

In differentiation of tumors, study group showed well differenciated category as the major subset, with a percentage of 46% followed by moderately and poorly differentiated varieties at 27% and 26% respectively, whereas L. P. Dragomir et al[1] group showed no clear distinction between the subsets, well differenciated constituting to about 37.28% followed by poorly and moderately differentiated at 35.59% and 27.11% respectively.

Patel MM et al[24] study has shown differentiation of tumours for well, moderate, and poorly differentiated varieties to be 60.12%, 38.7%, and 8.9% respectively.

Mehrotra Ravi et al[25] study, of the 303 malignant cases, the differentiation rates for well, moderate and poorly differentiated varieties are 56.76%, 41.91% and 1.3% respectively.

Khandekar et al[27] study has shown the rates of well, moderate and poorly differentiated varieties to be 33.75%, 20% and 18.75% respectively, with verrucous carcinoma at 27.5%.

Iype EM et al[26] has shown the rates of well, moderate and poorly differentiated varieties to be 52.6%, 34.2% and 4.2% respectively, undifferentiated being the rest.

Tumor progression

In tumor progression, study group showed that the subset invasive carcinoma with nodal metastasis constitutes 80% in contrast to L. P. Dragomir et al[1] group, in which, the cohort of invasive carcinoma without nodal metastasis had a percentage of 91%.

This is probably accountable to the late stage of presentation in the Indian sub continent.

Resection margins

In surgical cases, the number of negative margins in study group was 62% and in L. P. Dragomir et al[1] group, negative mergins were achieved in only 48% of cases.

The role of resection margins cannot be considered as the number of surgical cases in both the study and L. P. Dragomir et al[1] group is small to be statistically significant.

The site specific details of oral malignancies have been mentioned in the charts.

Out of 98 cases, eight cases were taken up for surgery, all of which were well differentiated.

Table 32 List of surgical cases

Lesion	Stage	Differentiation	Resection margins	Response	Adjuvant therapy	Recurrence
Tongue	I	Well	Involved	PR	RT	-
Lip	I	Well	Free	CR	-	-
Cheek	IVa	Well	Involved	PR	RT	+
Cheek	III	Well	Free	CR	-	-
Cheek	III	Well	Free	CR	-	-
Cheek	IVa	Well	Free	CR	-	-
Cheek/lip	IVa	Well	Free	CR	-	-
Floor of mouth	III	Well	Involved	CR	RT	-

Table 9 Treatment

No	Radiotherapy(RT)	Surgery(S)	Chemotherapy(CT)	Combined	Symptomatic
98	78	5	10	4 S + RT 3 CT + RT 1	1

Iype et al[26], in their study, have given the following treatment.

Table 33 Iype et al treatment protocol

Treatment	RT	Surgery	Surgery + RT	CT + RT	Palliative/no Rx	
No	96	21	45	38	43	264

RT as primary modality in 36.6% [96], RT followed by surgery in 8% [21] , CT and RT in 14.3% [38], surgery with adjuvant RT in 17% [45] and the lowest cohort of 8% [21] by surgery only.

A case of recurrent cancer was seen in a 55 year old male with cancer Rt cheek, stage Iva who underwent wide excision with forehead flap followed by RT for positive margins following resection. Recurrence was seen five months following RT.

A case of synchronous malignancy was seen in 71 year old male cancer Lt cheek/lip, stage IVa who underwent wide local excision with forehead flap. Patient had complete response.

Of the 10 cases who underwent chemotherapy, two cases had complete response- floor of the mouth cancer stage I and hard palate cancer stage II.

Of the four cases with combined modality treatment, one patient had complete response- floor of mouth stage III, who underwent wide local excision along with RT. Of the other cases, one patient had recurrence- carcinoma cheek stage IVa five months after surgery and RT and two had partial responses.

Risk factors

Tobacco and alcohol intake

Oral malignancies is a disease of middle aged and old aged male smokers. This has been evidently in the study group. The male preponderance in the study group is up to 73% and significant smoking history is associated in 57% of study group and tobacco chewing is seen in up to 50% of them. Tobacco chewing is associated in most of the women with oral malignancies to the tune of 100% and in about 47% males, mostly non smokers. Alcohol intake was seen in 24% of study group.

Smoking was not seen in the study group containing females.

In Iype et al study[26], 59.4% of patients had history of tobacco chewing or smoking or alcohol intake.

Khandekar et al[27] have shown in their study that tobacco chewing is seen in is seen in 71.3% and smoking in about 63.3%.

Other risk factors: No patients under study had STDs or HPV infection. Asssociations between risk factors were seen in 94% of study group and pre existing lesions in about 88%.

Tables 34 & 35 <u>Comparison between early [stages I and II] and late lesions [stages III and IV]</u>

Early lesions

CR			PR		
WD	MD	PD	WD	MD	PD
7	4	5	1	1	1
16			3		

Late lesions

CR			PR			No Response	Recurrence
WD	MD	PD	WD	MD	PD		
7	1	-	28	20	21	1 WD	1 WD
8			69			1	1

Tumour grade and tumour depth are important criteria for nodal metastasis[30]. But in the study group, 75% of poorly differentiated tumours had nodal metastasis in contrast to 80% in well and in moderately differentiated tumours.

In the study group, late stages of disease [stages III and IV] compromised up to 80% of study group, in contrast to 66.3% in Iype et al[26]. Nodal involvement was seen in 51.9% in Iype et al and in about 80% in the study group.

According to the study group, early lesions had more CR than late lesions.

Table 36 Response to treatment based on Differentiation

PD		MD		WD			
CR	PR	CR	PR	CR	PR	No response	Recurrence
5	22	5	21	14	29	1	1

The reasons for late presentation of oral malignancies has been explained by Sandeep Kumar et al[33] and the might hold good for the late presentation of the same in the study group.

The percentage of CR in well differentiated, moderately differentiated and poorly differentiated lesions is 31%, 19% and 19% respectively.

Table 29 **TREATMENT OUTCOME**

OUTCOME	NUMBER OF CASES	PERCENT
Complete relapse	24	24%
Partial relapse	72	73%
No response	1	1%
Recurrence	1	1%
Synchronous malignancy	1	1%

Table 37 **Sitewise Response to treatment** – Hard palate had more CR than other sites.

Site	CR	PR	No response	Recurrence	No
Lip	2	2	1	-	5
Cheek	**5**	30	1	1	36
Tongue	**4**	31	-	-	35
Hard palate	**10**	1	-	-	11
RMT	-	4	-	-	4
Gingiva	1	2	-	-	3
Floor of mouth	2	2	-	-	4

Prognostication in oral malignancies is based on stage and grade of the disease, of which stage is indirectly determined by

1. Size of the lesion
2. Involvement of adjacent structures [T3], which is resectable but chances of more chances of left over tumor
3. Involvement of base of skull, pterygoid plate, masticator space, or encasement of carotid artery [T4], which is inoperable
4. Presence or absence of lymph nodal/ distal metastasis[6,30]

Prognosis of oral malignancy is affected by the grade of tumors as nodal spread is common with moderately and poorly differentiated tumors, and the same has been demonstrated by the study group.[30]

Independently, tumour depth and grade are strong prognostic factors for nodal metastasis and tumour size and margins helps to predict local recurrences in the oral cavity independently of one another.[30]

Clinical factors in prognostication of oral malignancy

Table 38 Clinical factors in prognostication of oral malignancy

	Age		Sex		Lesion		Smoking			Alcohol		Tobacco	
	<49	>49	M	F	+	_	20p	+	-	+	-	+	-
Early lesion	5	14	16	3	13	6	11	4	4	4	15	12	7
Late lesion	16	63	56	23	74	5	44	11	24	19	60	38	41

26% of early lesions occurred in younger age group compared to 20% in late lesions.

Sex is not a risk factor for the development of oral cancer.[31] It has been considered here due to high incidence of oral cancers in males. In early lesions, up to 84% of cases is seen in males whereas in late lesions, it is seen only up to 70%. This can be probably explained by comparatively early medical attention seeking in males compared to females.

Pre existing lesions are seen in 68% in early lesions when compared to 94% in late lesions. Hence screening programmes if properly implemented may help to detect early lesions.

Significant smoking history is seen in 57% in early lesions and in 56% in late lesions.

Tobacco chewing is seen in 63% of early lesions to 48% of late lesions.

Alcohol intake history is seen in 21% of early lesions to 24% of late lesions. The low incidence of alcohol intake history is probably due to improper alcohol intake history given.

Table 39 Site wise prognostication – Hard palate had more early lesions.

	Early lesion	Late lesion	No
Lip	1	4	5
Cheek	1	35	36
Tongue	3	32	35
Hard palate	11	-	11
Retromolar trigone	1	3	4
Gingiva	1	2	3
Floor of mouth	1	3	4

As the study is a descriptive type of study, chi square test and P value could not be used to determine the significance of the study.

CONCLUSIONS

CONCLUSIONS

Oral malignancies is predominantly seen in males though sex is not a risk factor

Mainly affects old male smokers, but younger age of onset may be seen which should be thoroughly evaluated

Tobacco and alcohol consumption are two most important risk factors

Association between various risk factors in not only additive but also synergistic

Cheek and tongue have almost similar rates of involvement

Well differentiated SCC is the most common histological variety

Most common morphologic type being ulcerative

Size of the lesion and resection margins important prognostic factors in determining local recurrences

Grade of the tumour did not have a role in determining nodal spread in the study group

One case each of synchronous malignancy [cheek and lip] and recurrent cancer cheek, though statistically small can explain the concept of field cancerization

Early lesions have good response to treatment either in the form of RT or Surgery

Though stage of the disease is the major determinant of response to treatment independent of grade of tumour

Nodal disease has a bearing in the response rate and treatment option

More early lesions are found in males when compared to females probably due to early seeking of medical attention. Pre existing lesions were seen predominantly in the late lesions

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MASTER CHART

Guide to Master Chart

WD Well Differentiated

MD Moderately Differentiated

PD Poorly Differentiated

UP Ulceroproliferative

U Ulcerative

I Infiltrative

P Proliferative

Early – Early lesion – Stages I, II

Late – Late lesion - Stages III, IV

sl no	name	ip no	age	sex	socioecon	pre existi	smoking	alcoholism	tobacco	STD	size	LN	macroscop	biopsy	stage	diagnosis	treatment	response	e/l
1	mangalak	8882	50	M	low	+	30p	-	-	-	3*2	N1	UP	WD	III	floor of m	S + RT	Complete	late
2	tirikesh	13585	49	M	low	+	30p	1/d	-	-	1*1	N1	P	WD	III	angle of m	RT	Partial	late
3	perumal	15096	55	M	low	+	35p	1/d	-	-	3*2	N2c	I	MD	IVa	floor of m	RT	Partial	late
4	murugan	15311	50	M	low	-	25p	2/d	+	-	2*2	N0	UP	WD	I	hard palat	RT	Complete	early
5	balakrishr	15316	75	M	low	+	10p	-	+	-	3*2	N2c	UP	MD	IVa	Lt cheek	RT	Partial	late
6	natarajan	16204	74	M	low	+	20p	-	-	-	4*3	N1	UP	WD	III	Rt cheek	RT	Partial	late
7	arunachal	16205	62	M	low	-	35p	1/d	-	-	2*2	N1	U	PD	III	Lt RMT	RT	Partial	late
8	karpusam	16210	42	M	low	+	30p	-	+	-	1*2	N0	P	WD	I	Rt cheek	RT	Complete	early
9	rasaiah	16236	58	M	low	+	20p	1/d	-	-	4*3	N2c	U	WD	IVa	Lt tongue	RT	Partial	late
10	thiravium	18347	60	M	low	+	15p	-	+	-	3*2	N2a	U	MD	IVa	Rt tongue	RT	Partial	late
11	rathnam	18352	70	M	low	+	20p	-	+	-	5*4	N2a	UP	MD	IVa	Lt cheek	RT	Partial	late
12	sankaram	18359	65	F	low	+	-	-	+	-	3*2	N1	UP	WD	III	Rt cheek	RT	Partial	late
13	palavesan	18360	55	F	low	+	-	-	+	-	2*2	N1	UP	WD	III	lower lip	RT	Partial	late
14	chidamba	18374	57	M	low	+	25p	-	-	-	3*2	N1	I	WD	III	floor of m	RT	Partial	late
15	raiah	20807	36	M	low	-	15p	1/d	+	-	3*2	N1	UP	WD	III	Lt cheek	RT	Partial	late
16	murugan	20875	40	M	low	-	15p	-	+	-	2*1	N0	I	PD	I	hard palat	RT	Complete	early
17	thanuvel	22008	65	M	low	+	30p	1/d	-	-	3*1	N2b	P	PD	IVa	Rt cheek	RT	Partial	late
18	subraman	22268	45	M	low	+	10p	-	+	-	2*1	N0	U	WD	I	Lt tongue	RT	Complete	early
19	petchiam	23342	70	F	low	+	-	-	+	-	4*3	N2b	UP	WD	IVa	Rt cheek	RT	Partial	late
20	sivanamm	23333	55	F	low	+	-	-	+	-	3*2	N1	UP	WD	III	Lt cheek	RT	Partial	late
21	saravanan	25058	62	M	low	+	30p	-	-	-	3*2	N2a	UP	MD	IVa	Rt cheek	RT	Partial	late
22	poongani	27076	60	F	low	+	-	-	+	-	3*2(T4a)	N2b	I	PD	IVa	Lt cheek	RT	Partial	late
23	chellamm	28386	63	F	low	+	-	-	+	-	3*2	N2a	UP	WD	IVa	Rt cheek	RT	Partial	late
24	duraipand	28443	82	M	low	+	30p	-	+	-	2*1	N1	U	WD	III	Rt gingiva	RT	Partial	late
25	malaikani	29702	49	F	low	+	-	-	+	-	2*2	N1	U	MD	III	Lt tongue	RT	Partial	late

sl no	name	ip no	age	sex	socioecon	pre existi	smoking	alcoholisn	tobacco	STD	size	LN	macrosco	biopsy	stage	diagnosis	treatment	response	e/l
26	ganesan	29804	32	M	low	+	5p	-	+	-	2*2	N0	U	WD	I	Lt tongue	RT	Complete	early
27	poongani	30938	66	F	low	+	-	-	+	-	3*2	N2a	UP	WD	IVa	Lt cheek	RT	Partial	late
28	gurusamy	30947	70	M	low	+	30p	-	-	-	3*2	N2a	U	PD	IVa	Rt tongue	RT	Partial	late
29	saravanan	31152	62	M	low	-	25p	1/d	-	-	2*2	N0	I	MD	I	floor of m	CT	Complete	early
30	subban	15093	50	M	low	+	20p	2/d	-	-	3*2	N2c	U	PD	IVa	Lt tongue	RT+CT	Partial	late
31	saravanan	33258	60	F	low	+	-	-	+	-	3*2	N2c	UP	MD	IVa	Rt cheek	RT	Partial	late
32	mookan	34264	71	M	low	+	30p	-	-	-	4*3(T4a)	N2c	UP	WD	IVa	Lt cheek/l	S	Complete	late
33	malaikani	34477	55	M	low	+	-	-	+	-	3*2	N1	U	WD	III	Rt tongue	RT	Partial	late
34	aanai mut	34480	70	M	low	+	30p	1/d	-	-	3*2	N0	UP	PD	II	Lt gingiva	RT	Complete	early
35	pappa	38031	56	F	low	+	-	-	+	-	2*3	N1	I	PD	III	Lt RMT	RT	Partial	late
36	mariamma	38568	47	F	low	+	-	-	+	-	3*2	N2c	U	MD	IVa	Rt tongue	RT	Partial	late
37	mani	40165	70	M	low	+	20p	-	+	-	3*2	N0	I	MD	II	Lt RMT	RT	Partial	early
38	mani	40170	50	M	low	+	10p	-	+	-	3*3	N2b	I	MD	IVa	Rt tongue	RT	Partial	late
39	muthukur	42366	75	M	low	+	30p	-	-	-	3*2	N2b	U	MD	IVa	Lt tongue	RT	Partial	late
40	vadivel	42393	50	M	low	+	20p	-	-	-	3*2	N2a	UP	PD	IVa	Lt gingiva	RT	Partial	late
41	peramu	42822	70	F	low	+	-	-	+	-	3*3	N2a	UP	WD	IVa	Rt cheek	RT	Partial	late
42	thangasan	43585	48	M	low	+	20p	1/d	-	-	2*3	N2b	P	PD	IVa	Lt tongue	RT	Partial	late
43	mookayee	43626	36	F	low	-	-	-	+	-	2*3	N0	U	PD	II	hard palat	RT	Complete	early
44	raabith	43658	75	F	low	-	-	-	+	-	2*2	N0	U	PD	I	hard palat	RT	Complete	early
45	shanmuga	44817	60	M	low	+	25p	-	-	-	3*3	N2b	U	WD	IVa	Rt tongue	RT	Partial	late
46	sahul ham	44827	56	M	low	+	20p	-	-	-	4*3	N2a	UP	MD	IVa	Lt cheek	RT	Partial	late
47	valliamma	45276	60	F	low	+	-	-	+	-	4*3	N2b	UP	WD	IVa	Rt cheek	S	Complete	late
48	valli	46061	60	F	low	-	-	-	+	-	3*2	N0	I	PD	II	hard palat	RT	Complete	early
49	sivanamm	46789	71	F	low	+	-	-	+	-	3*2	N1	UP	WD	III	Rt cheek	S	Complete	late
50	velladurai	47076	55	M	low	+	30p	1/d	-	-	2*1	N1	UP	PD	III	rt angle of	RT	Complete	late

sl no	name	ip no	age	sex	socioecon	pre existir	smoking	alcoholism	tobacco	STD	size	LN	macroscop	biopsy	stage	diagnosis	treatment	response	e/l
51	valliappar	47078	75	M	low	+	30p	-	-	-	5*4(T4a)	N2b	UP	PD	IV a	Rt cheek	RT	Partial	late
52	chellaiah	48190	65	M	low	+	25p	1/d	-	-	4*3	N2a	UP	PD	IV a	Lt cheek	RT	Partial	late
53	pandi	48337	48	M	low	+	20p	1/d	-	-	3*2	N1	UP	MD	III	Rt tongue	RT	Partial	late
54	duraipand	49212	50	M	low	+	15p	-	+	-	3*2	N2a	I	MD	IV a	Lt tongue	RT	Partial	late
55	ganesh	49854	47	M	low	+	15p	-	+	-	3*2	N1	UP	MD	III	Rt cheek	RT	Partial	late
56	rajendran	51572	50	M	low	+	30p	1/d	-	-	3*2	N1	I	PD	III	Rt RMT	RT	Partial	late
57	thangakar	52652	60	F	low	-	-	-	+	-	2*2	N1	U	WD	III	lower lip	symptom	no respon	late
58	mathi	53205	55	M	low	+	30p	-	-	-	3*2	N1	UP	MD	III	Lt tongue	RT	Complete	late
59	sankaralir	54243	65	M	low	+	30p	-	-	-	2*2	N0	I	WD	I	hard palat	RT	Complete	early
60	dharmaraj	55557	45	M	low	+	20p	1/d	-	-	4*3	N2b	UP	WD	IV a	Rt cheek	RT	Partial	late
61	muthulaks	55796	52	F	low	+	-	-	+	-	3*2	N2a	UP	WD	IV a	Lt cheek	RT	Partial	late
62	esakkimut	56204	65	M	low	+	35p	1/d	-	-	2*1	N0	U	WD	I	upper lip	S	Complete	early
63	muthusan	57984	60	M	low	+	20p	-	-	-	3*2	N2a	UP	WD	IV a	Rt cheek	RT	Partial	late
64	arumugan	57999	67	M	low	+	30p	-	-	-	2*2	N2a	U	WD	IV a	Lt tongue	RT	Partial	late
65	arumugan	59272	67	M	low	+	15p	1/d	+	-	3*2	N1	U	WD	III	Rt tongue	RT	Partial	late
66	shanmuga	59714	50	M	low	+	30p	-	-	-	3*2	N1	UP	WD	III	Lt cheek	S	Complete	late
67	manikand	60427	42	M	low	+	20p	2/d	-	-	3*2	N1	U	WD	III	Rt tongue	RT	Partial	late
68	arumugan	60485	70	M	low	+	30p	-	-	-	3*2	N1	U	WD	III	Lt tongue	RT	Partial	late
69	pandi	60514	60	M	low	+	30p	1/d	-	-	3*2	N2a	U	WD	IV a	Rt tongue	RT	Partial	late
70	thangaiah	60515	60	M	low	+	20p	-	-	-	3*2	N1	U	WD	III	Lt tongue	RT	Partial	late
71	subbamm	217	75	F	low	+	-	-	+	-	3*3	N2a	U	WD	IV a	Rt cheek	RT	Partial	late
72	arjunan	1506	55	M	low	+	30p	-	-	-	3*2	N2a	U	WD	IV a	Lt tongue	RT	Partial	late
73	chellamm	1507	30	F	low	+	-	-	+	-	2*2	N1	I	WD	III	Rt tongue	RT	Partial	late
74	periyasam	1538	65	M	low	+	30p	-	-	-	3*2	N0	I	MD	II	hard palat	RT	Complete	early
75	ganapathi	2837	63	M	low	+	20p	-	+	-	3*2	N2a	U	WD	IV a	Lt tongue	RT	Partial	late

sl no	name	ip no	age	sex	socioecon	pre existir	smoking	alcoholism	tobacco	STD	size	LN	macroscop	biopsy	stage	diagnosis	treatment	response	e/l
76	utchimaha	3035	48	M	low	+	20p	-	-	-	3*3	N1	U	WD	III	Lt tongue	RT	Complete	late
77	bala singh	3956	37	M	low	+	-	-	+	-	4*3	N2c	UP	WD	IV a	Rt cheek	RT	Partial	late
78	pappa	4202	58	F	low	+	-	-	+	-	4*3	N2b	UP	MD	IV a	Rt cheek	RT	Partial	late
79	kadarkara	6748	65	M	low	+	30p	-	-	-	3*2	N0	I	MD	II	hard palat	RT	Complete	early
80	manikand	8825	42	M	low	+	10p	1/d	-	-	3*2	N1	U	MD	III	Rt tongue	RT	Partial	late
81	ramaiah	9761	72	M	low	+	30p	-	-	-	3*2	N0	I	PD	II	hard palat	RT	Complete	early
82	thangappa	10240	55	M	low	-	15p	1/d	+	-	4*3	N2c	P	WD	IV a	Rt cheek	S+RT®1R	Partial	late
83	chinnanac	11043	65	M	low	+	25p	-	-	-	4*3	N1	UP	MD	III	Rt cheek	RT	Partial	late
84	shanmuga	11068	50	M	low	+	15p	-	+	-	3*2	N2a	UP	PD	IV a	Rt cheek	RT	Partial	late
85	kumarasar	13827	68	M	low	+	30p	-	-	-	3*2	N2a	UP	PD	IV a	Rt cheek	RT	Partial	late
86	sivanaiah	15071	62	M	low	+	-	-	+	-	3*2	N1	U	PD	III	Lt tongue	RT	Partial	late
87	mariaselv	18746	36	F	low	+	-	-	+	-	2*1	N1	UP	PD	III	Lt cheek	RT	Partial	late
88	sudalaimu	22392	55	M	low	+	25p	1/d	-	-	4*3	N1	U	MD	III	Rt tongue	RT	Partial	late
89	susaiamm	27392	72	F	low	+	-	-	+	-	3*3	N2b	UP	PD	IV a	Lt cheek	CT	Partial	late
90	lingam	36975	40	M	low	+	20p	1/d	-	-	4*2	N1	U	PD	III	Rt tongue	CT	Partial	late
91	kalimuthu	39083	45	M	low	+	20p	-	-	-	3*2	N2b	U	MD	IV a	Lt tongue	CT	Partial	late
92	pathukanr	41721	57	F	low	+	-	-	+	-	4*2	N2c	UP	MD	IV a	Lt cheek	CT	Partial	late
93	subbaiah	43007	65	M	low	+	30p	-	-	-	2*2	N1	U	MD	III	Rt tongue	CT	Partial	late
94	natarajan	44607	53	M	low	+	15p	-	+	-	2*2	N0	I	WD	I	hard palat	CT	Partial	early
95	krishnasar	44757	70	M	low	+	30p	-	+	-	2*1.5	N0	U	WD	I	Rt tongue	S+RT	Partial	early
96	raj	46899	55	M	low	+	20p	-	+	-	3*2	N0	I	MD	II	hard palat	CT	Complete	early
97	muthamm	47048	70	F	low	-	-	-	+	-	2*2	N1	U	PD	III	Lt tongue	CT	Partial	late
98	madasamy	47078	70	M	low	+	30p	-	-	-	3*2	N1	U	PD	III	Rt tongue	CT	Partial	late

PROFORMA

Case No: IP No:

Name: DOA:

Age: Sex: DOD:

Address: Occupation:

SE status:

HISTORY

Swelling

Wound

Difficulty in swallowing

Excessive salivation

Loss of tooth

Spontaneous bleeding

Difficulty in mouth opening

Loss of weight and appetite

Any other swelling

H/o smoking- number of pack years

H/o alcoholism- number of drinks per day

H/o radiation exposure

H/o STD and AIDS

Pre-existing lesions

H/o similar lesions in the past

Family history

Co morbid illness

Previous surgeries

EXAMINATION

General examination

Build and nutrition/ BMI

Vitals

Pallor/ icterus/ cyanosis/ clubbing/ generalised lymphadenopathy

Hydration status

Examination of head and neck

1. **Upper jaw**
2. **Lower jaw**
3. **Temporomandibular joint**
4. **Oral cavity- lips/tongue/palate/gums/floor of mouth/cheek**
5. **Examination of salivary glands**

EXAMINATION OF REGIONAL LYMPH NODES:

VASCULAR EXAMINATION:

EXAMINATION OF FOR NERVE LESIONS:

SYSTEMIC EXAMINATION:

Cardiovascular system:

Respiratory system:

Abdominal examination:

INVESTIGATIONS:

Urine Albumin: Sugar:

Micro:

Blood- Hb% TC: DC: ESR:

Blood Sugar: Urea: Creatinine:

VDRL:

HIV

Culture and Sensitivity:

Biopsy:

X- rays

Ultrasound abdomen

CT scans

MANAGEMENT

- Surgical
- Radiotherapy
- Chemotherapy
- Combined
- Follow up